A DISSERTATION ON

RECONSTRUCTION OF PRESSURE SORES

MASTER OF CHIRURGIE

(M.Ch.,) Degree

BRANCH – III - PLASTIC SURGERY

THE TAMILNADU
DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

AUGUST 2009

DEPARTMENT OF PLASTIC SURGERY
MADURAI MEDICAL COLLEGE
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CERTIFICATE

This is to certify that this dissertation entitled "**RECONSTRUCTION OF PRESSURE SORES**" submitted by DR.M.SATHISH to the faculty of Plastic Surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement in the award of degree of MASTER OF CHIRURGIE IN PLASTIC SURGERY, Branch – III, for the August 2009 examination is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, Dr. M.SATHISH solemnly declare that the dissertation titled "**RECONSTRUCTION OF PRESSURE SORES**" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of MASTER OF CHIRURGIE, M.Ch., PLASTIC SURGERY, degree Examination to be held in AUGUST 2009.

Place : Madurai

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ACKNOWLEDGEMENT

I am deeply indebted to my beloved teacher Prof. Dr.C. BALASUBRAMANIAN, M.S., M.Ch., Professor and Head of Department of Plastic Surgery for his constant guidance, encouragement and untiring help throughout the period of this study.

I express my profound gratitude to Prof.V.DEVASENAN, Prof. V.NARAYANAN, Prof. A. CHARLES for their constant guidance and suggestions throughout my study period.

I also express my sincere gratitude to Dr. R.M. Raja Muthiah, Dr.S. Gnanasekaran, Dr.S.Aram, Dr. P.Suresh, Dr.V. Ravichandran, Dr.P.Parthasarathy for their valuable guidance and encouragement.

I thank the Dean, Madurai Medical College for permitting me to use the hospital facilities for my study.

I express my sincere thanks to all the PATIENTS, who inspite of their physical and mental sufferings, have co-operated and obliged to my request for regular follow up.

I express my thanks to my colleagues who helped me during my study.

Above all I owe my thanks to the ALMIGHTY for the successful completion of my study.

CONTENTS

INTRODUCTION

Pressure ulcers are localised areas of tissue necrosis that develop when soft tissue is compressed between a bony prominence and an external surface for prolonged periods of time. They have been reported throughout history (in the *Bible*, Lazarus, Job and Isaiah, among others, are thought to have had pressure ulcers) and in Egyptian mummies .

The terms *decubitus ulcer* and *pressure sore* have been interchanged inappropriately over the years. Technically, the term *decubitus ulcer* refers to wounds developed over bony prominences while in the recumbent position (ie, sacrum, heel, occiput); the Latin *decumbere* means “to lie down.” Therefore, semantically, wounds acquired from extended pressure in the seated or turned position (ie, ischial or trochanteric ulcers) are not decubitus ulcers. Therefore, in general, wounds acquired from pressure over bony prominences can always be called pressure sores.

While pressure is the main causative factor, many others – such as shear, friction, denervation, poor nutrition, age and smoking – can also contribute . Studies have suggested that, at any given time, 3-10% of hospitalized persons have pressure sores and 2.7% develop new pressure sores[(1)]. Among a selected population, the incidence rate for the development of a new pressure sore has been demonstrated to be much higher, with a range of 7-30%[(1)]. Overall, patients with pressure sores are important users of medical resources. They require 50% more nursing time, remain hospitalized for significantly longer periods, and incur higher hospital charges

Pressure ulcers appear in very ill patients and in states of prolonged immobilization. They are quite frequent in intensive care units and in paraplegic individuals. The expenses for their care are huge, due to the complicity of the long lasting treatment.

In 1938, Davis was the first to suggest replacing the unstable scar of a healed pressure sore with a flap of tissue [*(5)*]. In 1947, Kostrubala and Greeley recommended excising the bony prominence and adding padding for the exposed bone with local fascia or muscle-fascia flaps [(6)].

Shallow and superficial pressure ulcers are treated conservatively. Deep ones, with expressive underlying bone prominence in which no regression is on-going, are better to be treated operatively, if possible. Thus the hospitalisation period and the need for frequent dressings are shortened, preventing enormous scars and the risk of subsequent infection. Early and successful management of pressure sores ensures early rehabilitation of the patient.

AIM OF STUDY

The main objectives of this clinical study are

1. To evaluate the clinical results after a surgical reconstruction of pressure sores
2. To discuss the types,anatomical regions,planning and tecniques of reconstuctive methods.
3. To analyse the merits and demerits of individual reconstuctive option.
4. To identify aetiology and patient risk factors for pressure ulcers development.
5. To study the post operative management , risk factors for recurrance and complications
6. To formulate a reconstructive protocol for pressure sore, based on the outcomes of the study and existing literatue ,at the same time keeping in mind the resources available.

REVIEW OF LITERATURE

Pressure sore is a soft tissue injury resulting from unrelieved pressure over a bony prominence.

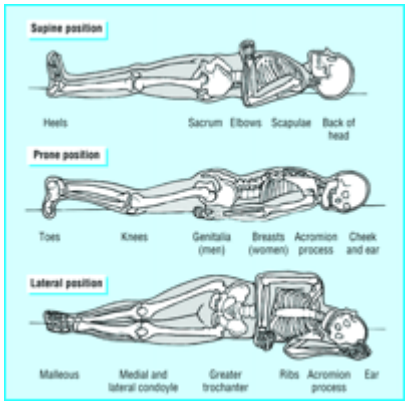
Aetiology

Several theories exist on the etiology of pressure sores, mostly based on ischemia and hypoxia resulting in decreased oxygen delivery to the tissues. In 1879, Charcot suggested that injury to CNS trophic centers decreases tissue tolerance to local pressure and leads to skin necrosis. However, Brown Sequard demonstrated that pressure ulcers can heal equally well in paralyzed and nonparalyzed animals.

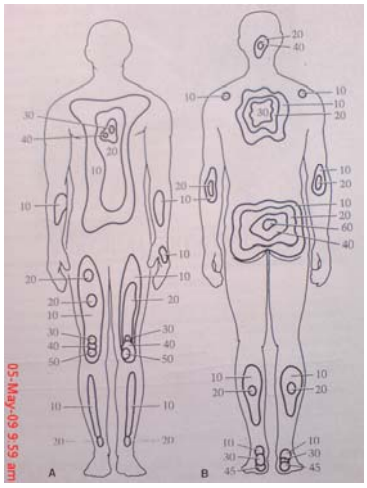
The pressure ischemia theory maintains that pressure sores result from constant pressure sufficient to impair local blood flow to soft tissue for an extended period. This external pressure must be greater than arterial capillary pressure of 32 mm Hg to impair inflow and greater than venous capillary closing pressure of 8-12 mm Hg to impede the return of flow for an extended time[(2)]. Constant external pressureof 70mmhg for 2 hours or more produces irreversible changes in tissues in animal model studies[(1)].

One study demonstrated no histologic changes with pressure release at 5-minute intervals.

Lindan et al documented ranges of pressure applied to various anatomic points in certain positions[8]. The points of greatest pressure with the patient supine included the sacrum, heel, and occiput, at 40-60 mm Hg[1,2]. With the body in prone position, the chest and knees absorbed the greatest pressure, at 50 mm Hg. When the patient is sitting, the ischial tuberosities are under the most pressure, at 100 mm Hg. Obviously, these pressures are greater than end capillary pressure, indicating why these are the most common areas to develop pressure sores.

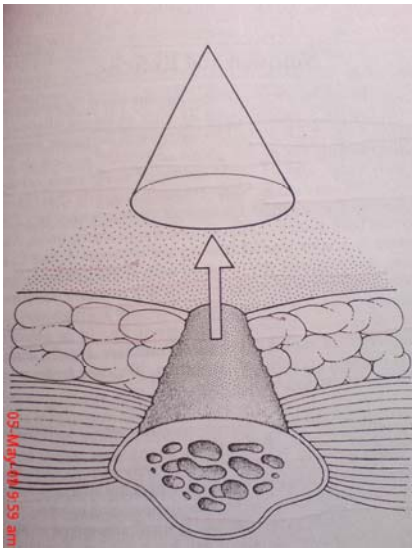


COMMON SITES OF PRESSURE ULCERS



DISTRIBUTION OF PRESSURE IN NORMAL HUMAN

Furthermore, studies have demonstrated the pathologic changes caused by pressure to be more severe in muscle than in skin and subcutaneous layers. These histologic studies revealed that early signs of damage occur in the upper dermis, with dilation of capillaries and venules and swelling and separation of endothelial cells. Then, perivascular infiltrates, platelet aggregates, and perivascular hemorrhage develop in the dermis. Additionally, subcutaneous fat demonstrates signs of necrosis along with early vascular changes. Interestingly, the epidermis shows no signs of necrosis until late because epidermal cells are able to withstand a prolonged absence of oxygen both in vivo and in vitro.



CONE SHAPED PATTERN OF INJURY

The molecular basis points to a imbalance between Matrixmettaloproteases (MMP) and Tissue inhibitors of metalloproteases (TIMP) as a causative factor[2]. Thus a cone shaped pattern of injury develops. The highest pressure and greatest injury is deep, adjacent to the bone. The cutaneous wound is only the tip of the iceberg.

Patient history

In obtaining a history from the patient with a pressure sore, the associated medical cause for the ulcer (eg, paraplegia, quadriplegia, spina bifida, immobilization in hospital, multiple sclerosis) must be determined. Other factors that should be elicited in the patient's history include onset, duration, other ulcers, prior medical treatment, wound care, and prior surgical treatment.

The patient's social situation also can impact treatment. Determine if the patient has a pressure-reducing mattress for the wheelchair and bed and an appropriate support system at home to minimize the risk of recurrence. Also, obtain a complete review of systems, including the presence of fevers, night sweats, rigors, weight loss, weakness, and loss of appetite. The risk factors for the development include, age, male gender,

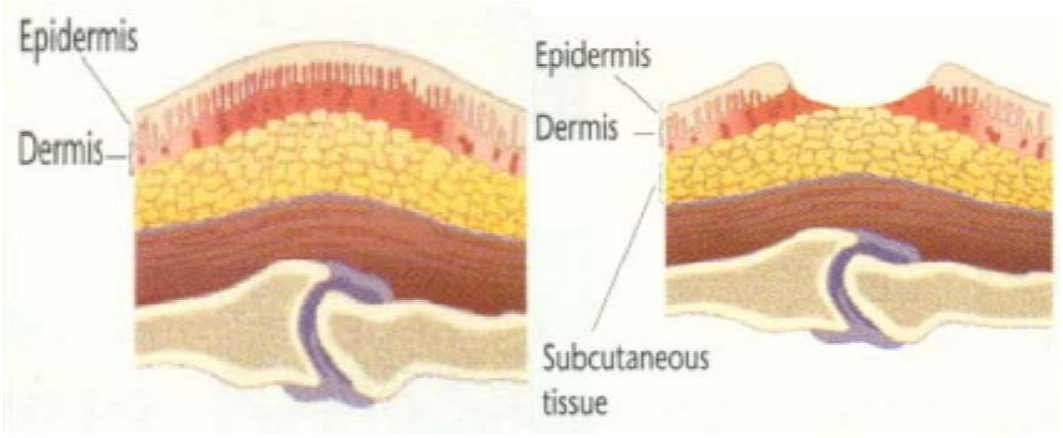
impaired sensory perception, moisture, immobility, poor nutrition, friction/shear, smoking, alcohol intake and immunosuppressants[2].

Physical examination

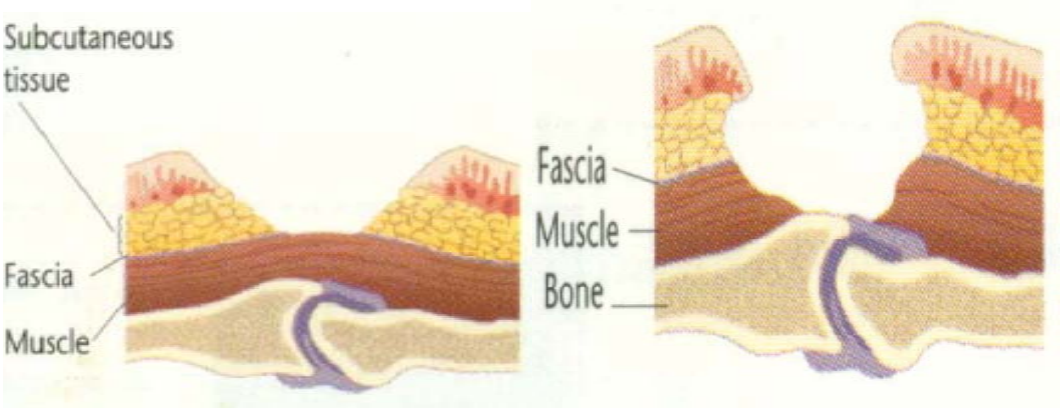
A thorough physical examination is performed describing the specific location of the pressure sore based on the underlying bony prominence (eg, sacral, ischial, trochanteric). 75% of pressure sores develop around the pelvic girdle with ischium, sacrum and trochanter being the commonest sites[2]. Infection of the pressure sore is suggested by wound edge erythema, foul odor, purulent discharge, and necrotic bone. The level of tissue injury (ie, to epidermis, dermis, subcutaneous fat, muscle, bone, joint)is determined. Several classification systems of pressure sores are available based on this level of injury. One widely accepted classification system has 4 stages. Pressure sore staging from Barczak et al[9] is as follows:

- Stage 1 - Skin intact but reddened for greater than 1 hour after relief of pressure
- Stage 2 - Blister or other break in dermis with or without infection
- Stage 3 - Subcutaneous destruction into muscle with or without infection
- Stage 4 - Involvement of bone or joint with or without infection

STAGE 1 STAGE 2



STAGE 3 STAGE 4



Also, note the character of the wound base and if it has granulation tissue or necrotic tissue. Verrucous heaps of white tissue within or around the wound suggest malignant transformation, as is observed with Marjolin ulcers. Document the size of the wound, wound edge undermining, additional pockets, and sinus tract communication with the hip joint or

urethra. Note existing scars and the presence of colostomy and cystostomy. Also assess the extent of associated spasm and contactures.

INDICATIONS

The decision to reconstruct a pressure ulcer is complex and based on several considerations. Stage 1 and 2 pressure sores are treated conservatively. In general, stage 3 and 4 pressure sores may require flap reconstruction, although some patients with stage 3 and 4 pressure sores must be treated conservatively because of coexisting medical problems.

Wound reconstruction can be considered once the bacterial load has been minimized to fewer than 100,000 organisms/gram of tissue, to reduce the risk of infectious complications[(1)]. Furthermore, the patient's social situation and nutritional status must be optimized (albumin level >3.5 g/mL),spasm controlled and contracture if present treated to reduce risk of an adverse outcome.

CONTRAINDICATIONS

Because the complication rate of pressure sore reconstruction can be extremely high, poor candidates for operations in general should not

undergo pressure reconstruction. Patients without the proper support network and pressure-release bed at home are not good candidates for pressure sore reconstruction because of the risk for recurrence or other complications. Patients who are noncompliant with nonoperative measures used to promote healing by secondary intention are also poor candidates for reconstruction.

Wound infections and osteomyelitis must first be aggressively debrided. Patients with significant fecal soiling into the pressure sore should be considered for diverting colostomy prior to reconstruction. Also, pressure sores with urethral fistulas should be diverted and healed prior to reconstruction.

INVESTIGATIONS

Lab Studies

- Quantitative culture by tissue biosy is taken, more then 100000 organisms per gram of tissue must be treated immediately.
- Osteomyelitis is suggested by an erythrocyte sedimentation rate (ESR) greater than 120 mm/h and a WBC count greater than 15,000/µL.

- Preoperative nutritional status can be determined by obtaining the patient's albumin level, which should be optimized to at least 3.5 g/mL prior to flap reconstruction. These patients often have anemia of chronic disease, suggested by a low mean corpuscular volume, and can be considered for a transfusion in order to achieve a preoperative hemoglobin level greater than 12 g.

Imaging Studies

- Diagnosis of underlying osteomyelitis can be evaluated first with plain films. Osteomyelitis can also suggested by positive bone scan findings. A negative bone scan finding generally excludes osteomyelitis[1]. However, patients with an open wound such as a pressure sore can often have a falsely positive bone scan. A positive bone scan finding can be evaluated further with an MRI or bone biopsy.

Diagnostic Procedures

- Bone biopsy is the criterion standard for the diagnosis of osteomyelitis within a pressure sore[1,2]. Bone biopsy should be

considered in patients with elevated ESR, elevated WBC, and or abnormal pelvic films suggestive of osteomyelitis.

Medical Therapy

Conservative treatment of pressure sores includes appropriate wound care, debridement of necrotic tissue, optimization of nutrition, release of pressure, and minimization of muscle spasticity to provide the patient with the best opportunity to heal by secondary intention. Stage 1 and 2 pressure sores are treated conservatively. Some patients with stage 3 and 4 pressure sores must be treated conservatively because of coexisting medical problems.

Infection of pressure ulcer wounds can affect treatment. To optimize wound healing potential, the wound should be in bacteriologic balance. Pressure ulcer infection is suggested by the presence of necrotic tissue, wound edge erythema, purulent discharge, and a foul odor. The most common organisms found in pressure sores include *Staphylococcus aureus, Proteus mirabilis, Pseudomonas aeruginosa, Bacteroides fragilis,* and *Bacteroides asaccharolyticus.* When more than 100,000 organisms/ gram of tissue, are growing on quantitative cultures obtained from the

pressure sore wound, debridement and aggressive wound care are required before considering wound reconstruction. Wound infection can be confirmed by quantitative tissue culture with more than 100,000 organisms in culture (clostridial and group B streptococcal infections can occur at lower bacterial counts).

For conservative treatment, remove all necrotic tissue. If necrotic tissue is minimal, moist-to-dry dressings can be used with modified Dakin solution or isotonic sodium chloride solution. More extensive necrosis may require surgical debridement at the bedside or in the operating room. Once the wound is clean, silver sulfadiazine can be used to reduce the bacterial load, which has been demonstrated to hasten wound healing. Alternatively, a negative pressure dressing can be considered for deep wounds that are clean and in bacteriologic balance. The negative pressure dressing has been found to decrease healing time and bacteria.

If the patient's nutritional status is deficient, as determined based on albumin level and caloric intake (energy intake), supplementation (either orally or enterically [tube feeding]) is required. In the short term, supplementation can be assessed by serum prealbumin level, which has a

shorter half-life (2 d) than albumin (17 d) or urine nitrogen. Patients with gut dysfunction may require total parenteral nutrition.

The pressure on the skin must be reduced by turning the patient in bed every 2 hours and by the use of a pressure-reducing mattress. Pressure-reducing mattresses include low–air loss beds, air-fluidized beds, and Roho cushion mattress seats for wheelchairs. Furthermore, sitting patients should shift their body weight every 15 minutes.

AIR – FLUIDISED MATTRESS

PRESSURE RELIEVING CUSHIONS

Surgical Therapy

In general, stage 3 and 4 pressure sores require flap reconstruction. The choice of flap for reconstruction depends on the location of the ulcer .

Preoperative Details

Involuntary muscle spasms must be controlled preoperatively with baclofen or diazepam. Arrange for a pressure-reducing mattress (air-fluidized bed) for the postoperative period. Treat the patient for urinary tract infection (UTI) if urinalysis and urinary culture findings (ie, nitrites, leukocyte esterase) confirm its presence.

Once the decision has been made to reconstruct, the wound is debrided. Debridement of a pressure sore that will be reconstructed is different from debridement of a pressure sore that will be treated conservatively (ie, allowed to heal by secondary intention). A radical bursectomy is performed by a Pseudotumor excision approach, placing a methylene blue–moistened sponge in the bursa and excising the pressure sore circumferentially, removing all granulation tissue, even from the wound base.

Anaesthesia

General anaesthesia is preferable to local or regional anaesthesia in the management of pressure sore. It avoids precipitating mass reflex, which may lead to respiratory paralysis and death. Autonomic nervous

system damage which may co-exist, can lead to hypotension, which may be aggravated by regional anaesthesia. General anesthesia gives good control of the respiratory and circulatory systems, and prevents the above complications.

Intraoperative Details

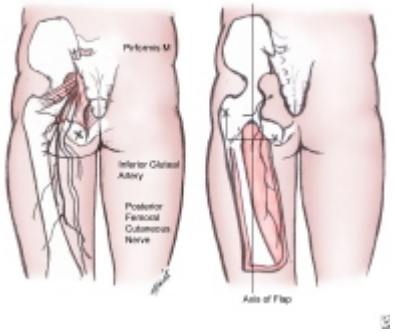
After the bursectomy, primary closure of the pressure sore is almost always under tension and is doomed to fail. Other technical points of pressure reconstruction include radically removing underlying necrotic bone, padding of the bone stump, filling the dead space with muscle, using a large flap, achieving adequate flap mobilization to avoid tension, and avoiding adjacent flap territories to preserve options to reconstruct other locations. The choice of flap for reconstruction depends on the location of the ulcer.

PSEUDO TUMOUR EXCISION

Ischial pressure ulcers

The ischial location is the most common location in individuals with paraplegia. When performing the excisional debridement in preparation for flap repair of ischial wounds, aggressive resection of the ischial tuberosity may risk a contralateral ischial pressure sore from increased contralateral pressure. Bilateral ischiectomy increases pressure on the perineum and increases the risk of perineal pressure sores[(1)]. Pressure sore recurrence is common in the ischial location. Therefore, the first option for reconstruction of ischial wounds is the gluteal thigh rotation flap, which does not preclude the future use of the inferior portion of the gluteus maximus muscle.

INFERIOR GLUTEAL THIGH FLAP



FLAP DESIGN AND MARKINGS

FLAP ELEVATION

The gluteal thigh rotation flap is an axial flap based on the inferior gluteal artery[3]. Both the biceps femoris and hamstring myocutaneous flaps transect the inferior gluteal artery. With the gluteal thigh flap, a superiorly based flap is elevated, with its axis being the inferior gluteal artery located between the greater trochanter and the ischial tuberosity . The gluteal thigh rotation flap is raised as a fasciocutaneous flap superiorly to the gluteal crease . The gluteal thigh flap may be raised to include the inferior portion of the gluteus maximus muscle, which increases the arc of rotation and also allows this flap to be used to reconstruct sacral defects .

Another popular option for ischial reconstruction, the inferior gluteus maximus myocutaneous flap, limits options for reconstruction of sacral wounds. Bilateral V-Y advancement flaps, inferiorly based random flaps, and superior gluteal myocutaneous flaps are not options for sacral reconstruction if an inferior gluteal myocutaneous flap has been used .

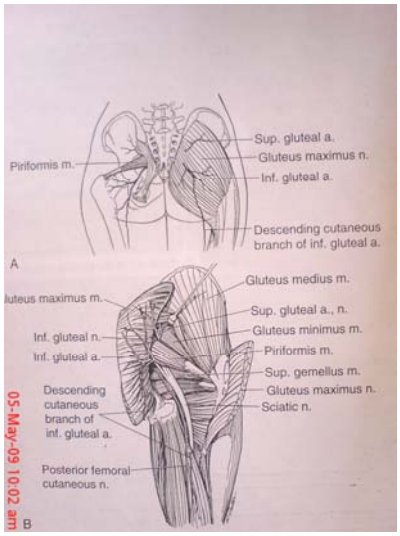
Other described options for ischial reconstruction include the hamstring myocutaneous flap, biceps femoris myocutaneous flap, tensor

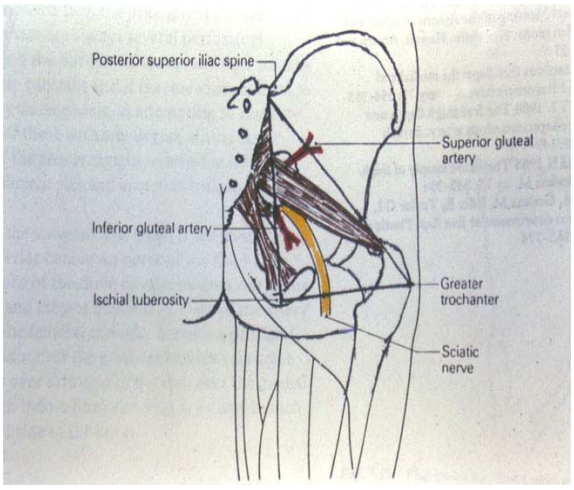
fascia lata (TFL) flap, gracilis myocutaneous flap, and medially based posterior thigh skin flap with or without biceps femoris.

Sacral pressure ulcers

Sacral ulcers are common in patients who have been on prolonged bed rest. Treatment involves complete ulcer excision, including the entire bursa, and conservative ostectomy. Small sacral ulcers can be reconstructed with an inferiorly based skin rotation flap with or without the superior gluteus maximus myocutaneous flap . The use of the random skin rotation flap does not preclude later use of the gluteus muscle. When using a random skin rotation flap, designing a large and wide flap with an axis of rotation that permits tension-free closure is essential.

ANATOMICAL STRUCTURES IN GLUTEAL REGION

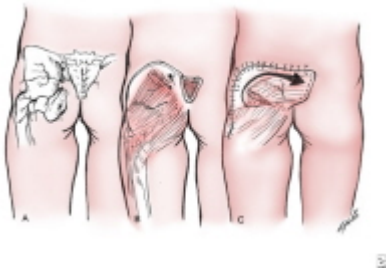




With the superior gluteal myocutaneous flap, a wide skin rotation flap is elevated with the superior portion of the gluteus maximus muscle. The landmarks for the superior gluteal artery on which the superior gluteus maximus muscle flap is based include the posterior superior iliac spine (PSIS) and the ischial tuberosity. The superior and inferior gluteal arteries branch from the internal iliac artery superior and inferior to the piriformis muscle approximately 5 cm from the medial edge of the origin of the gluteus maximus muscle from the sacrococcygeal line (from the PSIS to the coccyx[(3)].

When using the superior portion of the gluteus maximus muscle as a flap, it is elevated in a lateral-to-medial direction to avoid injury to the superior gluteal artery, which can be difficult to identify from the medial direction because of the inflammation and scar of the sacral pressure sore. The insertion of the superior portion of the gluteus maximus muscle is the iliotibial; this insertion is released. The superior gluteal artery is only 4 cm long, which limits the rotation of the muscle. Thus, harvesting the entire length of the muscle may be necessary to allow for rotation or turnover into the defect without tension.

GLUTEUS MAXIMIUS MYOCUTANEOUS FLAP



ROTATION DESIGN V-Y SLIDING ADVANCEMENT

Larger sacral ulcers require the use of bilateral flaps such as bilateral V-Y myocutaneous advancement flaps[3]. V-Y flaps can be based on the superior, inferior, or whole gluteus maximus muscle depending on ulcer location. The V should be fashioned wide enough and long enough to close as a Y without tension. The medial edge of the origin of the gluteus maximus is elevated in a medial-to-lateral direction for approximately 4 cm because the superior and inferior gluteal arteries enter the gluteus muscle 5 cm from its origin.

Release of the gluteal muscle insertion laterally is important for medial advancement and tension-free approximation of the muscles medially. Inflamed fibrous tissue along the medial muscle edge can be preserved and used to hold sutures for midline muscle approximation. Another option for sacral reconstruction is the transverse lumbosacral

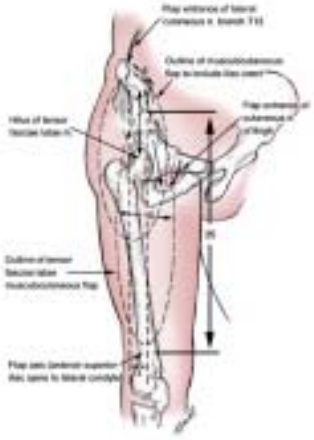
flap, based on the ipsilateral lumbar perforators notably L3, located at the lateral border of the paraspinal muscles.

Trochanteric pressure ulcers

Trochanteric pressure sores are less common and are typically associated with minimal skin loss. Excisional debridement of trochanteric ulcers in preparation for flap repair involves resection of the entire bursa and greater trochanter of the femur. The TFL flap is the first option for reconstruction of trochanteric pressure sores[(3)]. The TFL flap is a myocutaneous flap based on the lateral femoral circumflex artery. The TFL muscle is 13 cm long, 3 cm wide, and 2 cm thick, and it originates from the anterior superior iliac spine (ASIS) and the iliac crest and inserts into the iliotibial tract.

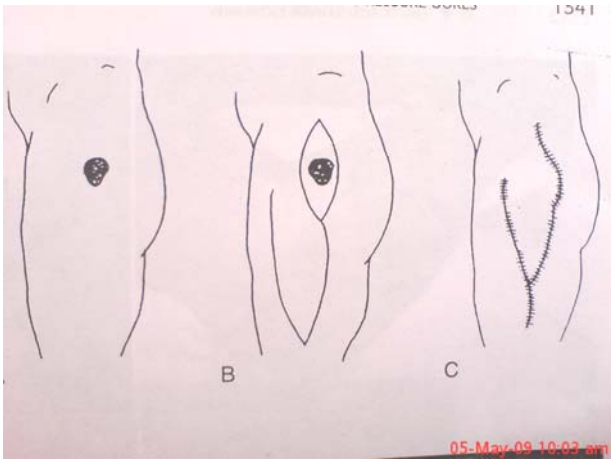
The skin paddle is harvested 10 cm in width and designed over the muscle along an axis from the ASIS to the lateral tibial condyle . The inferior limit of the cutaneous territory can be extended to 6 cm above the knee and 25-35 cm in length. The lateral femoral circumflex artery can be found approximately 6-8 cm inferior to the ASIS. In patients with lumbar

lesions, a sensate TFL flap can be designed to include the T12 dermatome by fashioning the flap to include the area 6 cm posterior to the ASIS.



TENSOR FASCIA LATA FLAP – FLAP DESIGN AND MARKINGS

Other described modifications of the TFL flap include the retroposition V-Y flap and the bipedicled TFL. Other options for trochanteric reconstruction include the vastus lateralis myocutaneous flap, the gluteal thigh flap, and the anterior thigh flap.



TFL FLAP – V-Y ADVANCEMENT

Multiple pressure ulcers

Multiple pressure sores can be observed in the same patient. Reconstruction of multiple ulcers may require the use of a total thigh flap[3]. The total thigh flap is a long and formidable operation necessitating the transfusion of 6-20 units of blood. It should be reserved as a salvage procedure when other attempts have been unsuccessful. Patients who have undergone a unilateral total thigh flap can sit a wheelchair.

Free tissue transfer, either muscle or fasciocutaneous flaps can be considered if no options to use locoregional tissues are available. Perforator based and propeller flaps are recent additions to the surgical options and can be performed for a small to medium sized pressure sore.

Postoperative Management

Postoperative care of pressure sore reconstruction is extremely important to reduce the risk of complications. From the time of transfer from the operating table to the air-fluid bed, exercise care to prevent shearing and tension across the flap repair. Patients are positioned flat in the air-fluid bed for 4 weeks. After 4 weeks, the patient can be placed

carefully into a semi-sitting position. Six weeks after surgery, sitting is begun initially for 10-minute intervals. After these sitting periods, evaluate the patient's flap for discoloration and wound edge separation. The sitting periods are increased at 10-minute intervals over 2 weeks for up to 2-hour periods. Patients are taught to lift themselves to relieve pressure for 10 seconds every 10 minutes

COMPLICATIONS

Pressure sores are associated with a number of adverse outcomes. Complications include autonomic dysreflexia, osteomyelitis, pyarthroses, sepsis, amyloidosis, anemia, recurrence, urethral fistula, and malignant transformation.

Autonomic dysreflexia is a disordered autonomic response to specific stimuli. Patients with midthoracic spinal cord lesions are most prone to this response, which includes sweating, flushing, nasal congestion, headache, intermittent hypertension, piloerection, and bradytachycardia. The sweating and flushing occur proximal to the level of injury. Patients in whom autonomic dysreflexia is suggested are first

positioned with their head up and are monitored for changes in heart rate and blood pressure. Then, the precipitating stimulus must be removed.

The most common precipitating cause is bladder distension, which requires treatment with Foley catheter insertion or irrigation of an existing Foley catheter to remove blockage. Consider a rectal examination to evaluate for fecal impaction. Nifedipine, hydralazine, or topical nitroglycerin can be used to stabilize the blood pressure. Finally, spinal anesthesia may be required for autonomic dysreflexia refractory to the above measures.

Foremost in the treatment of osteomyelitis is the removal of all nonviable bone, down to bone that bleeds bright red. When reconstructing pressure sores associated with osteomyelitis, use bone that is in the base flaps and has a muscle component. Place the muscle over this bone after appropriate bone debridement. The flap reconstruction can be performed at the same time as the bone debridement. Then, administer a 6-week course of intravenous antibiotics.

Pyarthrosis of the hip joint can occur with communication of ischial or trochanteric ulcers. Often, the femur head contains osteomyelitis,

which mandates its removal. The Girdlestone arthroplasty procedure has been described for this situation (ie, hip pyarthrosis), with removal of the femur head and reconstruction of this space with the vastus lateralis muscle flap[(11)].

Recurrence rates for pressure sores can be high because of patient noncompliance, seromas, hematomas, wound infections, and dehiscence. Intraoperative measures that can minimize recurrence risk include tension-free flap reconstruction, suction catheter drainage, meticulous hemostasis, and aggressive debridement. Postoperatively, patients must use pressure-reducing mattresses and pressure-release techniques to decrease their risk of recurrence. Patients with paraplegia have the highest rate of recurrence (80%).

Pressure sores can also erode into the urethra. Treatment of this complication (ie, urethral fistula) involves urinary diversion. Pressure sore reconstruction can be considered once the fistula has healed .

Although Marjolin initially described malignant transformation of a chronic scar from a burn wound, the term *Marjolin ulcer* has been used interchangeably for malignant transformation of any chronic wound,

including pressure sores, osteomyelitis, venous stasis ulcers, urethral fistulas, anal fistulas, and other traumatic wounds. This malignant transformation is, histologically, a well-differentiated squamous cell carcinoma; however, its behavior is very aggressive when it arises in pressure sores as compared to in burns or osteomyelitis. Pressure sore carcinoma is a highly lethal disease, with 12 of 18 patients reported in the literature dying within 2 years. Therefore, treatment of a pressure sore carcinoma must be aggressive.

Wide local excision, amputation, and lymph node dissection have been described for Marjolin ulcers arising from burns or osteomyelitis. However, because pressure sore carcinoma is more aggressive, more radical treatment is required (eg, hemicorporectomy and regional node dissection) if a cure is to be effected[(2)]. In a retrospective analysis at M.D. Anderson Cancer Center, in 3.4% of 1200 patients with squamous cell carcinoma, the carcinoma arose from thermal burns of irradiation dermatitis. The actual complication rate for malignant transformation of a pressure sore is not known but can be assumed to be low because only 18 cases are described in the literature.

The most common causes of fatality for patients with chronic pressure sores are renal failure , pneumonia, septicemia and amyloidosis. In general, mortality rates are higher for patients who develop a new sore and in whom the pressure sore fails to heal.

OUTCOME AND PROGNOSIS

With the highest complication rate of all procedures performed, pressure sore reconstruction is perhaps the most complex challenge to plastic surgeons. Treatment of patients with pressure sores involves several considerations to minimize the risk of adverse outcomes of the reconstruction. Patients must be meticulously and compulsively prepared preoperatively, with nutritional deficiency, anemia, spasms, and coexisting urinary infection corrected. Patients must have adequate social resources, including pressure-release beds, wheelchair mattresses, and a compliant attitude to prevent recurrence. Intraoperatively, technical points of pressure sore reconstruction must be followed stringently to minimize the risk of complications. The postoperative regimen for the transition from flat bed rest to sitting and from weight-shifting into and out of the wheelchair in the return to daily living must be strict and careful.

Even with close adherence to the above stated guidelines, pressure sore recurrence rates are high. When caring for patients with chronic pressure sores, flap procedures must be planned carefully and social resources must be provided assiduously to reduce the high risk of adverse outcomes in this complication-prone population

FUTURE AND CONTROVERSIES

Wound healing factors that promote spontaneous wound closure of pressure ulcers have been identified. Platelets play an important role in the inflammatory phase of wound healing by providing hemostasis and releasing growth factors known to accelerate wound healing. Atri et al reported their success in augmenting spontaneous wound healing with platelet-derived growth factors for 23 subjects in whom initial treatment with Silvadene failed[12]. Later, Robson et al and Mustoe and colleagues found recombinant platelet-derived growth factor BB in concentrations of 100 mcg/mL to effect clinical wound healing in a phase II study of stage 3 and 4 pressure ulcers[13,14].

The cost to treat patients with these growth factors is presently significant. However, in the future, these nonoperative measures may be involved in the treatment of pressure ulcers, especially for patients who are not suitable for surgery because of coexisting morbidities.

MATERIALS AND METHODS

Materials

This work includes the study of 50 patients who underwent reconstruction for pressure sores at the Department of Plastic surgery,Government Rajaji Hospital, Madurai.

The patients who were admitted Orthopaedics, Neurology, Neurosurgery, General Medicine and General Surgery wards and subsequently referred to Plastic Surgery at Government Rajaji Hospital, Madurai, were studied between October 2006 – April 2009.

Methods

The methods include obtainiing history from patients, thorough clinical examination and necessary investigations and appropriate surgical reconstruction. Intraoperative, post operative complications were noted and managed accordingly. Patients were advised regarding rehabilitaion and referred back to their respective departments and advised regular follow up. The patients were followed up every week for two month, then monthly for a period of six months. The maximum follow up was for a period of 6 months.

All informations were entered in a proforma specially designed for this study.

Methodology

The patient's name,age, sex, history of presenting illness and its duration was obtained. Past history of chronic medical and surgical illness noted. Personal history like smoking, alcohol consumption and diet pattern were obtained.

Detailed physical examination of the pressure sore was made and tissue diagnosis was recorded and reconstruction planned accordingly.Neurological examination regarding sensory, motor impairment, bladder, bowel control, presence of spasms and contractures were noted.

Basic investigations like blood Hb estimation, urine examination, blood sugar and renal parameters like urea, creatinine were done. Serum protein levels were assessed. Wound Swabs for culture and sensitivity were taken.

X ray chest, X ray of the local part and ECG were taken. Hypoprotenemia was managed by appropriate nutritional supplementation. Infection was controlled by periodic debridement and

antibiotics. Spasm relieved with Diazepam 5mg twice daily. Adequate relief of pressure was obtained by change of position every 2 hours, avoidance of moisture and nursing in a water bed.

All the patients were informed about the surgical procedures,the intraoperative, post operative complications and rehabilitation. A detailed informed consent regarding the procedure and its complications was obtained. Patients were operated under general anaesthesia

Postoperatively all the patients were managed until suture removal. Blood transfusion was given if indicated. If necessary patients were observed in the intensive respiratory unit for a couple of days. Patients were advised regarding rehabilitaion and referred back to their respective departments and advised regular follow up.

The patients were followed up every week for two month, then monthly for a period of six months. The maximum follow up was for a period of 6 months.

ANALYSIS

The data obtained was analysed for the following factors

1. Age and sex of the patient
2. The primary aetiology (T8 and above – high spinal, T9 and below- low spinal)
3. Blood Hb level
4. Serum Albumin level
5. Presence of infection
6. Stage of the pressure sore
7. Size of the pressure sore (Small <5cm., Medium 5-10cm., Large >10cm.)
8. Site of the pressure sore
9. Reconstructive method
10. Complications (Haematoma, Infection, Dehiscence, Necrosis, Recurrance)

Factors associated with pressure ulcer development and the outcomes of surgical management were analysed and the results were obtained.

OBSERVATION AND RESULTS

In the fifty pateints included in this study,the mean age was 46.36 years,with a range of 16- 80 years.

Table I

Age and gender

Age / Gender	< 20	21-40	41-60	60-80	Total
Male	1	12	16	7	36
Female	2	4	4	4	14
Total	3	16	20	11	50

72 % patients were male and 28% were female in our study.

Female to male ratio was 1: 2.57

Table II

PRIMARY DIAGNOSIS

Primary Diagnosis	No. of cases	%
Traumatic Paraplegia – low spinal level	20	40
Traumatic paraplegia – high spinal level	4	8
Traumatic quadriplegia	6	12
Tuberculous paraplegia low spinal level	2	4
Tuberculous paraplegia high spinal level	1	2
Tumour compression –paraplegia low spinal level	1	2
Tumour compression – paraplegia high spinal level	2	4
Fracture neck of femur	3	6
Fracture Pelvis	3	6
Head Injury	5	10
Encephalitis	3	6

Traumatic Paraplegia low spinal level (T9 and below) was found to be the aetiology in 20 patients. Post traumatic paraplegia high spinal level and Post traumatic quadriplegia were present in 4 and 6 patients respectively. Tuberculosis and Tumour compression causing paraplegia

were found in 3 patients each. Orthopaedic injuries like fracture neck of femur and fracture pelvis were the causative factors in 3 patients each. Pressure sore development due to alteration of conscious level were found in 8 patients.

Table III

Risk Factors for Pressure Sore development

Infection was present in 80 % of all the pressure sores, which required serial debridement, antibiotic therapy and periodic dressings before attempting reconstruction. Anaemia and Hypoalbuminaemia were present in 35 and 33 patients respectively . Anaemia was treated with Iron supplements and blood transfusion wherever necessay. High protein diet was recommended for all patients with low albumin levels.

Risk Factors	**No. of cases**	**%**
Hb < 10 gm%	35	70
Serum Albumin < 3 gm%	33	66
Presence of Infection	40	80

Table IV

ANATOMICAL SITE OF PRESSURE SORE

Anatomical Site	No of cases	%
Sacrum	34	68
Ischium	10	20
Trochanter	6	12
Patella	1	2
Lateral Malleolus	1	2

The commonest Site was the sacrum, which was present in 34 patients, followed by Ischium and Trochanter in 10 and 6 patients respectively. Pressure sore of the patella and lateral malleolus was seen in one patient each, in combination with Trochantric sore.

Table - V

SIZE OF THE PRESSURE SORE

Size	No. of cases	%
Small (<5cm.)	14	28
Medium (5-10cm.)	21	42
Large (>10 cm.)	15	30

Medium sized pressure sore was noted in 42 % of patients. 14 patients had small pressure sores and remaining 15 had large pressure ulcers.

Table VI

STAGE OF PRESSURE SORE

Stage	No. of cases	%
Stage 2	6	12
Stage 3	14	28
Stage 4	30	60

The majority of patients, 30 in number, had a Stage 4 pressure sore. Stage 3 sores were found in 28% of patients. Only 6 patients requiring reconstruction had stage 2 ulcers.

Table VII

METHOD OF RECONSTRUCTION

Reconstruction	No. of cases	%
B/L VY Gluteus Maximus Myocutaneous flap	7	14
Gluteus maximus rotation flap	3	6
B/L VY Skin advancement flap	1	2
Skin rotation flap	4	8
B/L Skin rotation flap	5	10
Limberg flap	12	24
Double Z Rhomboid flap	2	4
Transverse Lumbosacral back flap	2	4
Inferior Gluteal thigh flap	6	12
Tensor Fascia Lata flap	6	12
Secondary suturing	2	4
Split skin graft	2	4

Sacral pressure sore reconstrution was performed with B/L VY Gluteus maximus myocutaneous flap in 7 patients and B/L rotation flap in 5 patients.The majority of the Ischial pressure sore were reconstruted with Inferior gluteal thigh fasciocutaneous flap, accounting 6 patients. Tensor Fascia lata flap was used for Trochantric sores in 6 patients. Limberg flaps were used in all sites of pressure sores to a total of 12 patients.

Table – VIII

Complicatons

Complications	No. of cases	%
Haematoma	6	12
Infection	5	10
Wound Dehiscence	6	12
Flap Necrosis	3	6
Recurrance	5	10

Haematoma and wound dehiscence were the commonest complications, encountered each in 6 patients. The infection rate was found to be 10%, while flap necrosis, which were partial were found in 3 patients. In our 6 month follow up recurrance rate was found to be 10%.

CLINICAL PHOTOGRAPHS

SCARAL PRESSURE SORE

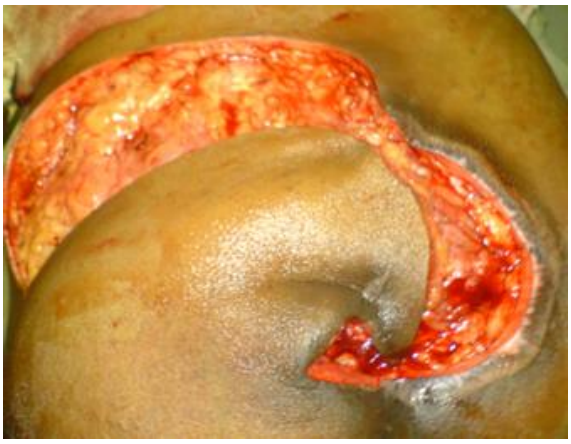
V-Y GLUTEUS MAXIMUS SLIDING ADVANCEMENT FLAP

SCARAL PRESSURE SORE

LIMBERG FLAP

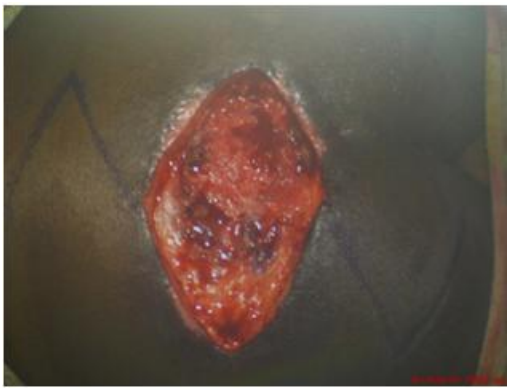
SCARAL PRESSURE SORE

GLUTEUS MAXIMUS ROTATION FLAP



SCARAL PRESSURE SORE

DOUBLE Z RHOMBOID



SCARAL PRESSURE SORE

ROTATION SKIN FLAP

TRANSVERSE LUMBOSACRAL BACK FLAP

SCARAL PRESSURE SORE

B/L SKIN ROTATION FLAP

SCARAL PRESSURE SORE

B/L SKIN ROTATION FLAP

ISCHIAL PRESSURE SORE

INFERIOR GLUTEAL THIGH FLAP

ISCHIAL PRESSURE SORE

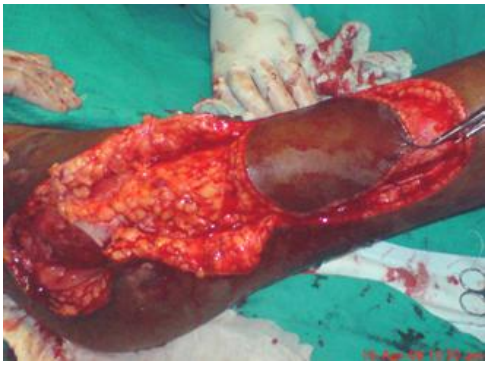
FASCIOCUTANEOUS ROTATION FLAP

TROCHANTRIC PRESSURE SORE

TENSOR FASCIA LATA FLAP

TROCHANTRIC PRESSURE SORE

TENSOR FASCIA LATA ISLAND FLAP



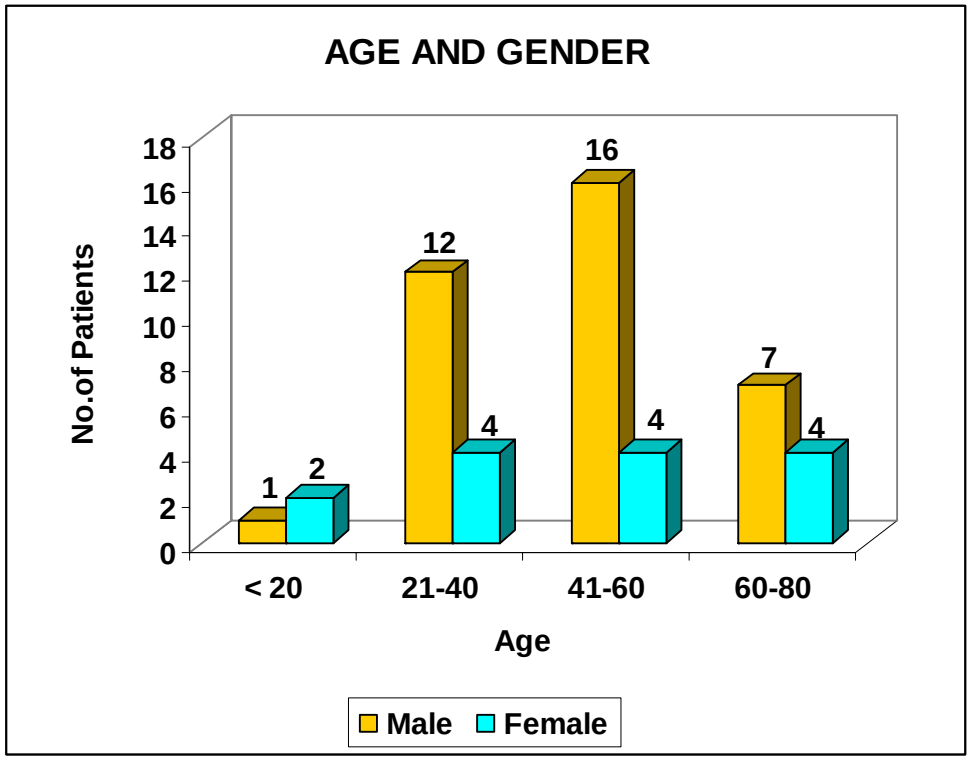
TROCHANTRIC PRESSURE SORE

LIMBERG FLAP COVER

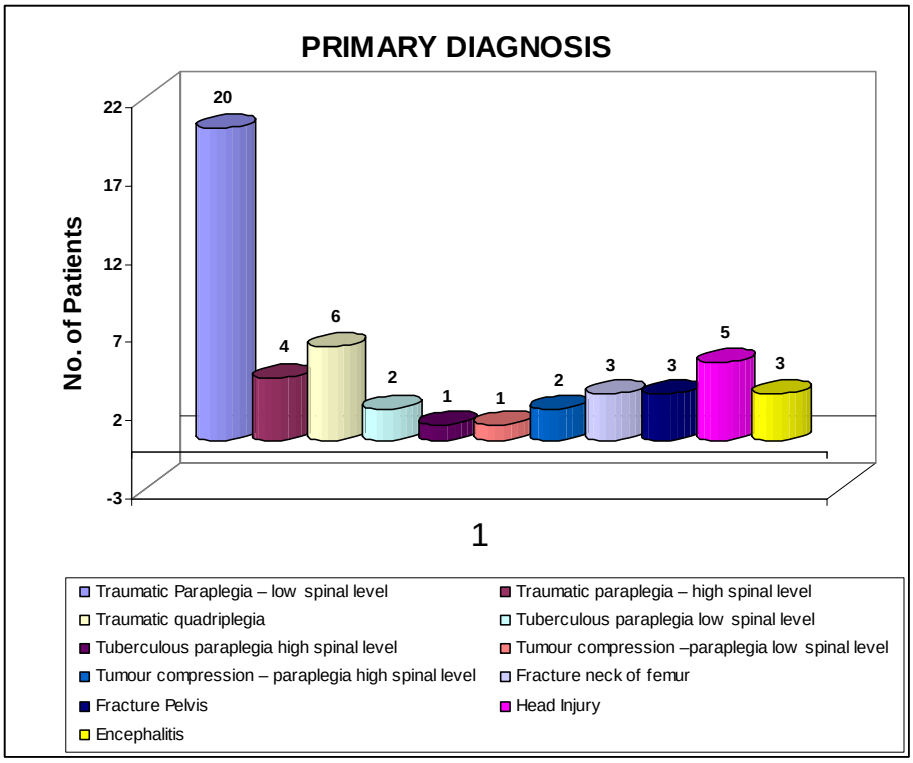
DISCUSSION

Reconstruction of pressure sore is performed to achieve early healing in order to continue the rehabilitation process and treatment of primary illness which resulted in pressure ulcer.

In our study of 50 patients, requiring reconstruction, the majority of the patients, 36 out of 50 patients were in 20-60 age group, which is the most productive period of an individual. Men were twice more commonly involved than females. This may be due to the fact that more men developed the primary neurological dysfunction resulting in pressure ulcer.



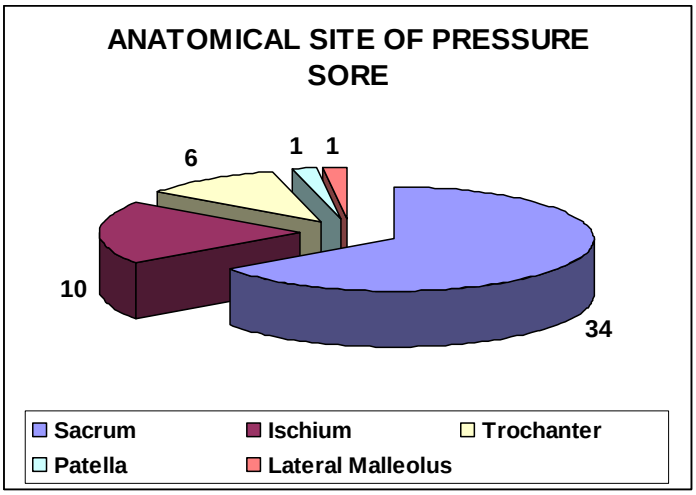
Traumatic etiology was found to be the cause in more than 80% of patients. Most of the trauma resulted from road traffic accidents. Infectious aetiology namely encephalitis & tuberculosis accounted for only 12% of the patients. Patients developed anemia and hypoalbuminaemia either due to the injury or subsequently, which when added to poor nursing care, increases the chance of ischemia and subsequent development of pressure ulcers. Once a stage I pressure sore develops, the wound gets infected due to the presence of deep seated devitalised tissue and this becomes responsible for the progression of pressure sore in depth and size.

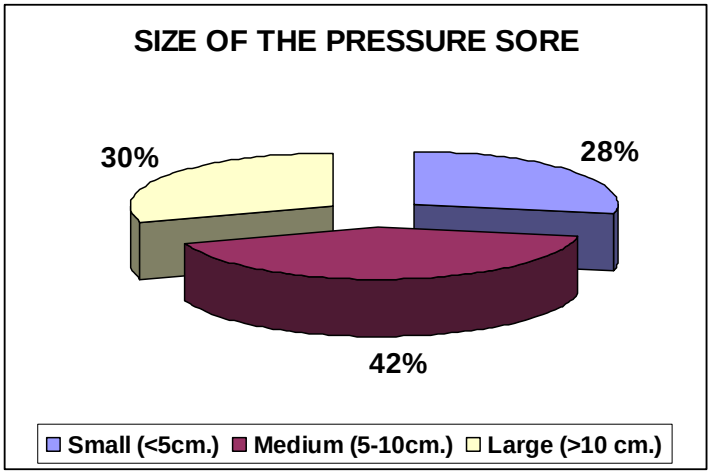


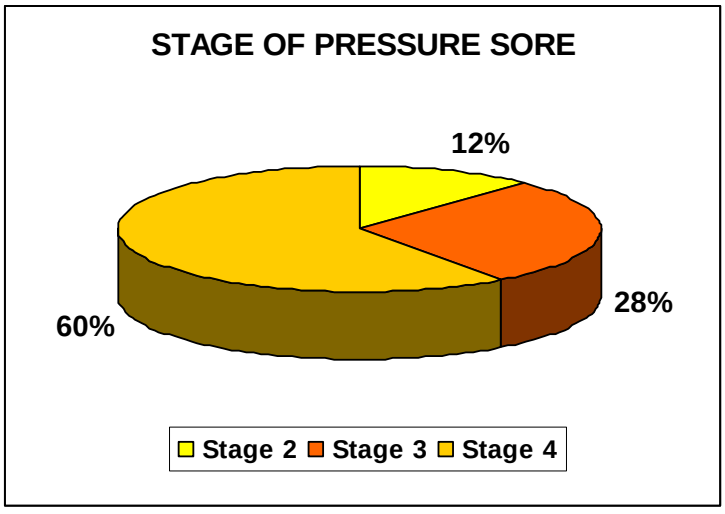
Hence effective control in incidence of RTA plays an important role in primary prevention. Management of anaemia, hypoalbuminaemia with control of infection plays a prime role in secondary prevention of pressure ulcers.

In our study most of the patietns 68%, developed sacral pressure sore, which is in contrast with western literature, where most common site is ischium[1,2]. Ischial pressure sore were seen only in patients who had signs of recovery and regained some motor function, so that they are able to sit in a chair. Trochantric pressure sores were predominantly seen in patients with high spinal injuries, who developed spasm and contractures of hip joint, resulting in lying down in lateral positions.

Stage I and most of stage II pressure sores when managed conservatively, healed well and did not warrant reconstruction. Around 90% of the reconstruction were done only for stage III and stage IV pressure sores. The number of patients were almost equal in all the three categories relating to the size of the pressure sore, with size 5-10cms being the most common to get reconstructed.


ANATOMICAL SITE OF PRESSURE SORE
6
1
1
10
34
Sacrum
Ischium
Trochanter
Patella
Lateral Malleolus


SIZE OF THE PRESSURE SORE
30%
28%
42%
Small (<5cm.)
Medium (5-10cm.)
Large (>10 cm.)


STAGE OF PRESSURE SORE
12%
28%
60%
Stage 2
Stage 3
Stage 4

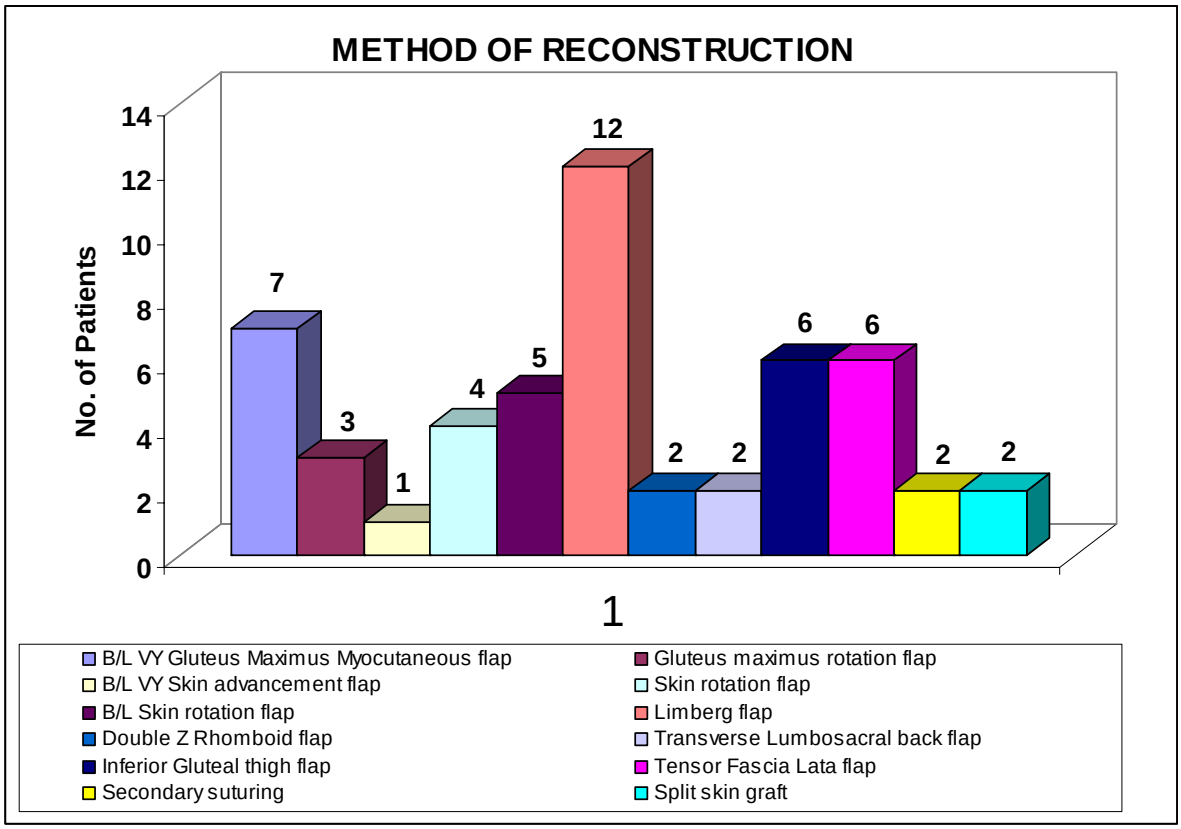
A through understanding of the principles and options of surgery allows the optimal procedure to be performed. In general all pressure sores needs debridement in the pseudotumour approach as discussed earlier. Ostectomy is always done for a trochantric sore, done partial for an ischial sore and very minimal for a sacral sore.

Sacral pressure sore that are stage IV are better managed by a Gluteus maximus myocutaneous flap. It can be a rotation gluteus maximus for a small to medium midline ulcer, unilateral or bilateral V-Y advancement for a medium to large pressure ulcer. Superficial sacral sores can be reconstructed with skin flaps. The commonest flaps used was the limberg flap. It gives good results for a small sacral sore. When the size is large limberg flap may lead to wound dehiscence. Hence a double Z rhomboid design can be used, which evenly distributes the tension around the raw area. Rotation skin flaps and Transverse lumbosacral back flap are also viable options for sacral pressure sore management.

Ischial sores are best managed by a Inferior Gluteal thigh flap. Since ischial sores are prone for recurrence, in case of future need, this flap does not jeopardize blood supply of other flaps. Other options include

a gluteus maximus myocutaneous flap based on inferior gluteal artery and rotation skin flaps.

Tensor fascia lata is the flap of choice for a trochantric sore. It can be pedicled or islanded. Limberg flap can also be used for small ulcers. In using a limberg flap for pressure sore, we have to keep in mind to plan the flap in such a way that it does not affect blood supply of other flaps in the vicinity.



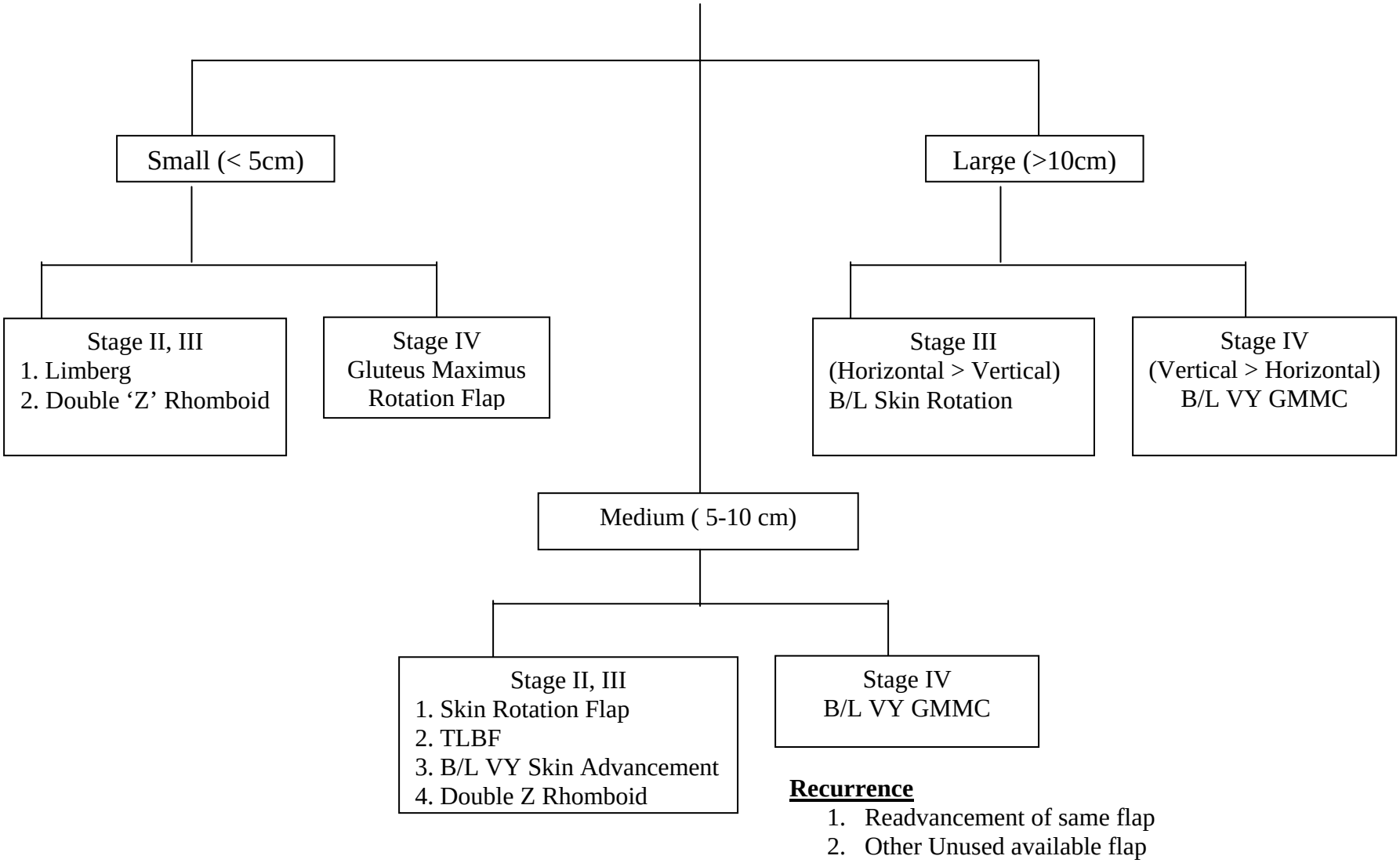
In our study, haematoma leading on to wound infection and dehiscence was found to be around 10%. This was more common in muscle flaps and TFL flap, where there is more dissection and dead space. A closed suction drain and perfect haemostasis is a must in the reconstruction of pressure sores. Partial flap necrosis was found more with skin flaps, which were salvaged and managed conservatively.

Even with optimal preoperative, efficient intraoperative, good post operative management and rehabilitation, pressure sores are prone for early or late recurrence. In our study recurrence rate was 10% for a 6 month follow up period.

This confirms the time tested fact that for pressure sores, prevention is better than cure.

RECONSTRUCTION PROTOCOL

SACRAL PRESSURE SORE

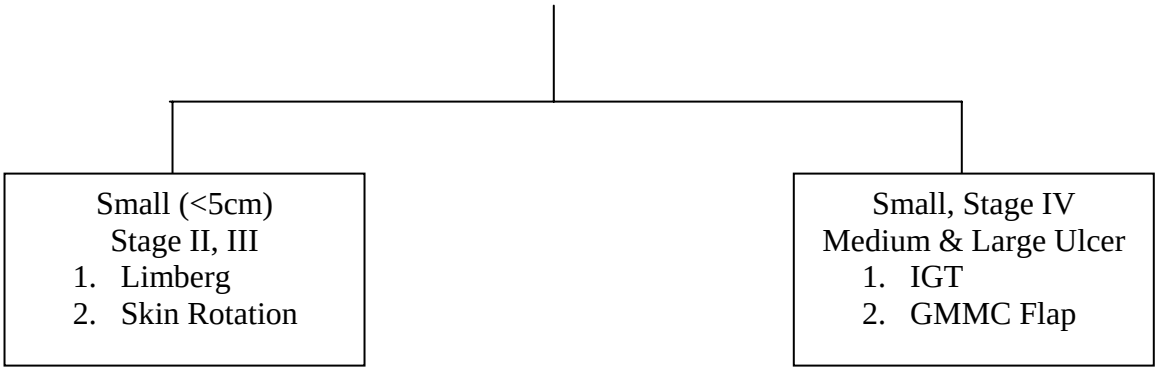


Recurrence

1. Readvancement of same flap
2. Other Unused available flap

RECONSTRUCTION PROTOCOL

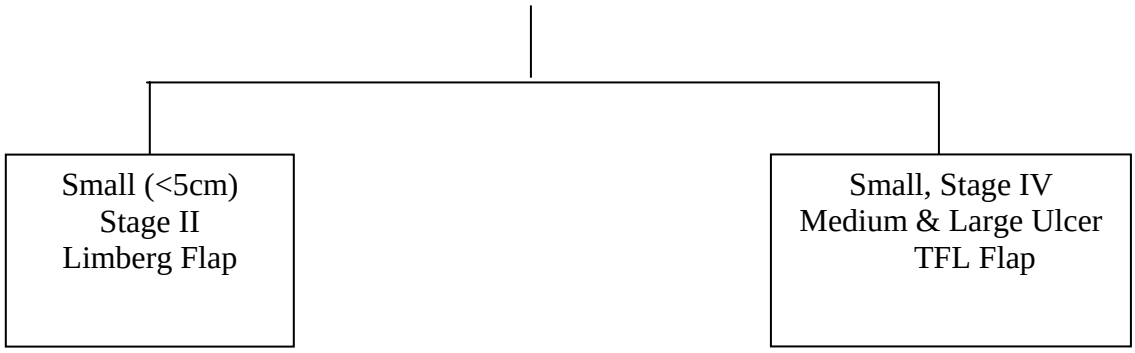
ISCHIAL PRESSURE SORE



Recurrence

1) Gracilis Myocutaneous Flap
2) V-Y Hamstring Myocutaneous Flap

TROCHANTRIC PRESSURE SORE



Recurrence

1) IGT Flap
2) Groin Flap

CONCLUSION

In Conclusion

1. Sacral Pressure sores of Stage III and IV are best managed by one of the variants of Gluteus maximus myocutaneous flaps. Skin flaps can be used to reconstruct superficial Sacral sores.
2. Tensor fascia lata myocutaneous flap and Inferior gluteal thigh fasciocutaneous flap are reliable reconstructive options for Trochantric and Ischial sores respectively.
3. Effective prevention and management of anaemia and hypoalbuminaemia in addition to good nursing care reduces the incidence of pressure sores. Effective control of Infection by medical and surgical means in early stages, prevents progression of pressure sores, and promotes early healing.
4. Trauma was the primary causative factor, leading to neurological damage in majority of patients, and prevention and control of road traffic accidents is of prime importance in primary prevention of pressure sores.

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DEPT OF PLASTIC, HAND, RECONSTRUCTIVE &

MICROVASCULAR SURGERY.

GOVERNMENT RAJAJI HOSPITAL, MADURAI MEDICAL COLLEGE

RECONSTRUCTION IN PRESSURE SORE

Name: Ward :

Age : Address:

Sex :

Occupation:

I.P. No.

D.O.A PRIMARYDIAGNOSIS

D.O.S

D.O.D

Primary complaint

History of present illness

- duration
- discharge
- fever
- bladder and bowel continence

Past & Personal H/ O

- Diabetes/ Hypertension/ Smoking / Peripheral Vascular disease/ Alcohol intake, Similar pressure sores in past, previous surgery

General Examination:

- Anaemia / Avitaminosis
- pedal edema

Investigations:

1. Hb %
2. TC,DC,ESR
3. Urine – Albumin,Sugar, Deposits
4. Wound Swab
5. Serum Proteins, Albumin
6. X-ray of local part

Local Examination

Site

Size

Stage

Infection/ Slough

Surrounding Skin

Neurological Examination

Higher Functions, Conscious level

Sensory level

Motror level

Spasm

contractures

Reconstuctive method:

Complications :

Post operative follow up:

Rehabilitation :